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Highlights of Heart Progress; Symposium on Chemical Carcinogenesis; Dental Caries and the Pediatrician

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Highlights of Heart Progress—1961

Public Health Service Publication No. 949, pp. 15–21.

Dietary Surveys

Much of the evidence linking dietary fat to atherosclerosis in humans has been derived from epidemiological studies. These compare carefully selected population groups, which differ sharply in their mortality from atherosclerosis, but which are as similar as possible in all other respects except for the dietary factors under study. In this way, it is hoped, the specific dietary factor or factors responsible for the mortality difference can be effectively isolated from among the many other variables that might also operate in atherosclerosis.

Such studies have accumulated enough impressive evidence against cholesterol and saturated fats to make them prime suspects as the chief dietary contributors to atherosclerosis. Conversely, a large body of evidence has accumulated to suggest that unsaturated fats, especially such polyunsaturated fatty acids as linolenic and arachidonic acids, lower serum cholesterol levels, and it has been postulated that they may confer some measure of protection against atherosclerosis.

Some examples:

South and East African Negroes, who subsist on a diet very low in total fat, animal fat, and protein, have a much lower mortality from heart disease than do South African white settlers, whose dietary patterns—and mortality from heart disease—are similar to those of Americans.

Atherosclerosis appears to be almost nonexistent among young Negroes of these groups but does occur in middle-aged and older Negroes. The disease is much less severe and its lesions contain less lipid than in the case of Americans. Most striking, however, is the extreme rarity of myocardial infarction ("heart attacks") and other cardiovascular disorders involving blood clots, even among those Negroes with atherosclerosis.

High-fat diets appear to increase the tendency of the blood to clot and also to impede the fibrinolytic mechanisms by which the body dissolves clots. Thus, the low-fat diet of the African Negro may confer protection against heart disease not only by retarding the development of atherosclerosis but also by preventing the thromboembolic complications usually attending the disease and often the immediate cause of its crippling and lethal effects.

Many other studies on selected populations from all over the world have revealed the same striking associations between diet (especially dietary fat), serum cholesterol levels, and relative proneness or immunity to atherosclerosis. Among the factors studied have been total dietary fat, animal (saturated) versus vegetable (chiefly unsaturated) fat, and the protein-fat-carbohydrate ratio of the diet.

That dietary factors, not genetic factors, may be primarily responsible for the differing degrees of susceptibility to atherosclerosis exhibited by these populations has been suggested by other studies. These compared population groups from countries with a low incidence of heart disease with groups of the same racial origin who had migrated to this country and adopted "Westernized" dietary patterns.

The studies have shown that the low mortality from heart disease apparently enjoyed by some of the natives of Italy, Japan, Africa, and other countries is not shared by their racial counterparts in America who succumb to heart disease with about the same distressing frequency as do other Americans.

If diet is largely responsible for high death rates from cardiovascular disease, then the possibility exists that changing our habitual dietary patterns might reduce our presently better-than-even odds of dying from one of this family of diseases.

A proposal now under consideration involves a series of grant-supported feasibility studies to determine whether it is possible and desirable to undertake a large-scale study to elucidate precisely the relationship between the kind of fat in the diet and mortality from coronary heart disease.

For these preliminary studies, a dietary regimen has been tentatively worked out which involves a large reduction of the proportion of animal fat in the diet and the substitution of unsaturated vegetable oils. The regimen would be simple enough to be followed easily in the average home and, hopefully, palatable enough to be followed voluntarily by large groups for long periods of time.

Animal Studies

Studies on the effects of diet on atherosclerosis in laboratory animals are complicated by the fact that few commonly used laboratory animals develop the disease spontaneously. In most, atherosclerosis can be induced experimentally only by diets extremely high in cholesterol, and frequently this diet must be augmented by drugs that interfere with normal fat metabolism or by surgically produced hypertension. The lesions produced may differ from those of humans and are rendered somewhat suspect by the drastic methods required to produce them.

Primate colonies, such as those established this year, should contribute greatly toward the solution of this problem. Primates, physiologically, are very similar to man. Furthermore, many primates appear to develop atherosclerosis spontaneously, with lesions closely resembling those of the disease in humans. Thus, these animals should be ideal for more extensive, more detailed laboratory studies of important leads uncovered by epidemiological research.

One such study, comparing Kenya baboons with baboons from a primate colony, has shown that both groups develop atherosclerosis spontaneously even though their average serum cholesterol level (114 mg percent) is only about half that of humans (225 mg percent).

However, their serum also showed a decided preponderance of low-density lipoproteins, which transport most of the cholesterol in serum, and, in humans, such a preponderance is associated with proneness to atherosclerosis. The relationships observed between the development of atherosclerosis in baboons and such factors as age, sex, and diet also closely paralleled those observed in epidemiological studies on human populations.

Studies on Fat Metabolism

Studies in this area have been aimed at clarifying the mechanisms governing the absorption, synthesis, transport, and breakdown of fatty substances in the body. If, as many believe, atherosclerosis is a disease caused primarily by abnormal lipid metabolism, basic studies of this sort are the ones most likely to extend medical control over atherosclerosis.

Cholesterol

The cholesterol consumed in the normal human diet is probably the least important source of the cholesterol of serum. The major source is the liver, which readily synthesizes cholesterol from acetate, a two-carbon building block arising from the metabolism of protein and carbohydrate as well as from fat.

The liver can produce enough cholesterol to maintain excessive serum cholesterol even when the diet contains none. In fact, excessive serum cholesterol levels have been induced in animals by diets containing little fat of any kind, but containing excessive calories derived from carbohydrates. Other studies have indicated that the total caloric content of the diet may be at least as important a factor in its effects on serum cholesterol as the amount and kinds of fat it contains.

Studies on the mechanisms regulating cholesterol production by the liver indicate that cholesterol synthesis is controlled by a double feedback system. Feedback, one of the most important mechanisms for regulating the synthesis of any compound by the body, prevents enzyme systems from glutting the market with their particular product, almost literally, by drowning the enzyme system in its own surplus.

If a substance is made at a faster rate than it can be used or gotten rid of, the surplus that accumulates interferes with further production, usually by inhibiting an enzyme involved in some earlier step in the production line. The surplus product is often so similar in chemical structure to the substance ordinarily processed by that enzyme (called the substrate) that the enzyme cannot discriminate between the two. Thus, the product is able to compete with the substrate for the enzyme's active site.

The enzyme cannot act on the surplus product perched on its active site, but, as long as it is there, the enzyme also cannot act on its normal substrate. As more and more surplus product accumulates, it increasingly crowds out the enzyme's normal substrate and slows further production.

A surplus of cholesterol apparently inhibits two earlier steps in the complex sequence of reactions by which cholesterol is made from acetate. A diet high in cholesterol can almost completely suppress cholesterol synthesis in many laboratory animals, but this inhibition appears to be less pronounced in man. Cholesterol synthesis appears to account for 60–75 percent of the serum cholesterol in humans, even when their diet is high in cholesterol.

Another feedback mechanism affecting cholesterol synthesis involves bile acids, the major pathway for cholesterol excretion. The accumulation of bile acids blocks some step in their production from cholesterol. If this cholesterol cannot be diverted into other metabolic channels, it piles up, inhibiting further cholesterol synthesis.

A number of hormones and drugs affect serum cholesterol levels primarily by affecting the relative rates of cholesterol synthesis and excretion. For example, thyroid hormones lower serum cholesterol even though they actually step up the rate of cholesterol synthesis. Serum cholesterol falls because these hormones cause an even sharper increase in the production and excretion of bile acids.

Serum cholesterol levels may also be rather precisely determined by genetic mechanisms, other factors being equal. One study on these genetic factors compared serum cholesterol levels in a group of identical twins (who presumably carry the same genes) with these levels in a similar group of fraternal twins, who are genetically distinct as any two children of the same parents.

Although the group as a whole exhibited a wide range of serum cholesterol patterns, when each pair of twins was considered separately, the serum cholesterol pattern of one identical twin always corresponded very closely with that of the other. The differences in serum cholesterol patterns between fraternal twins were much greater.

The mechanisms by which saturated fats act to increase serum cholesterol and by which unsaturated fats act to reduce it are still obscure. Unsaturated fats actually appear to increase cholesterol synthesis. Like the thyroid hormones they might overcompensate by stepping up bile acid excretion, but proof for such a mechanism is lacking. Over 70 percent of serum cholesterol is combined with fatty acids (cholesterol esters). One hypothesis suggests that esters in which cholesterol is combined with unsaturated fatty acids may be more readily taken up from the circulation by the tissues.

Unsaturated fats will not lower serum cholesterol when superimposed on a diet high in saturated fats. They must be *substituted* for these saturated fats. The balance between unsaturated and saturated fats in diets designed to reduce serum cholesterol appears to be a very delicate one. One such diet, containing 30 percent of its calories as unsaturated fats and 8 percent as saturated fats reduced serum cholesterol levels by 15 percent. However, the addition of only 1 percent of saturated fat to this diet completely eliminated its cholesterol-lowering effect.

Lipoproteins

Lipoproteins are large fat-protein molecular complexes synthesized by the liver. They appear to be the transport vehicles for about 95 percent of the total serum lipids. (The other 5 percent is free fatty acids, which are transported by albumin.) For convenience, lipoproteins are divided somewhat arbitrarily into two weight classes: high-density and low-density lipoproteins. Each class can carry any sort of lipid except free fatty acids, but, as it turns out, the low-density fraction usually carries the major portion of cholesterol and neutral fat (triglyceride) of serum.

The rate of lipoproteins synthesis and the ratio of high-density to low-density lipoproteins are affected by genetic, nutritional, and hormonal factors. It is still an open question whether the ratio of lipoproteins affects the handling of fat by the body or whether the shoe is on the other foot, with the amount and kind of fat available determining the lipoprotein ratio.

In any case, a preponderance of low-density lipoproteins is usually associated with high serum cholesterol and relative proneness to atherosclerosis, whereas a robust high-density fraction appears to confer some measure of protection against the disease.

The Yemenite Jews, who appear to be virtually free from atherosclerosis and heart disease, have a relative preponderance of high-density lipoproteins that appear to carry a large proportion of their serum cholesterol, which is low. When diabetic Yemenites were placed on the typical diabetic diet—high in fat and protein, low in carbohydrate—they tended to develop lipoprotein patterns similar to those of Europeans.

The immunity to atherosclerosis enjoyed by the Yemenites appears to reside more in their large high-density lipoprotein fraction than in their low serum cholesterol. This was suggested by studies comparing Yemenites with other Jews who had coronary heart disease but did not have high serum cholesterol levels. In fact, some of these had serum cholesterol levels as low as those of healthy Yemenites, but all of the coronary patients had much lower values for serum high-density lipoproteins.

The female sex hormones (estrogens) that protect the premenopausal woman against atherosclerosis do so primarily by increasing the relative amount of serum lipid carried by the high-density lipoproteins. It is also noteworthy that diets which reduce serum cholesterol levels also appear to reduce the proportion of low-density to high-density lipoproteins in many cases.

Tangier disease, a rare familial disorder of lipid storage discovered only last year, is providing unique opportunities to study the role of the two lipoprotein classes in human fat metabolism. The disorder is characterized by the accumulation of extremely large quantities of cholesterol in reticuloendothelial tissues, such as those of the tonsils, lymph nodes, liver, and spleen; by abnormally low serum cholesterol levels and by the virtual absence of serum high-density lipoproteins. Tangier disease provides the first reported instances of a deficiency of this type of lipoprotein.

The clinical manifestations of Tangier disease indicate that high-density lipoproteins may be essential to the normal uptake of cholesterol by tissues and may also play a role in its esterification. Studies on this disorder are continuing at NIH, and, when correlated with similar studies on recently discovered patients who lack low-density lipoproteins, may help to clarify the roles played by the two lipoprotein classes in human fat metabolism.

The Atherosclerotic Lesion

Other important studies have concentrated on the mechanisms by which the lipids of atherosclerotic lesions are deposited in the blood vessel wall. It has not yet been determined conclusively whether these lipids diffuse passively into the wall from the serum, are produced locally by the blood vessel tissues themselves, or are deposited at the site of mechanical injury or clot formation.

Each of these theories has its staunch advocates and its supporting evidence. Too, there appears to be no natural law against all three processes getting their licks in at one stage of the disease or another.

The filtration theory holds that lipoproteins carrying cholesterol or other lipids diffuse passively into the arterial wall, become trapped in the maze of fibrous tissue, and gradually accumulate to form lesions.

Studies on the aortas of dogs with experimentally produced atherosclerosis disclosed a definite gradient of lipid deposition along the length of the aorta. Cholesterol deposition was highest in the ascending aorta and progressively less down its length in the early stages of atherosclerotic development.

Aortic specimens studied later in the course of the disease revealed highest concentrations of cholesterol in the abdominal aorta. This is believed to be caused by the slower rate at which cholesterol leaves this site after diffusing passively into the arterial wall.

Autopsy studies on human aortas exhibiting a full range of atherosclerotic lesions have suggested that these lesions may develop as follows:

The first stage is the appearance of fatty streaks, composed of lipids synthesized and extruded by cells of the arterial wall. These fatty streaks were found in aortas from all autopsy subjects over three years old, but their rate of development appeared to be most rapid about the time of puberty. The fatty streaks were followed about twenty years later by the appearance of fibrous plaques. The fatty streaks appeared to be reversible, but the fibrosis did not.

Other studies indicate that some atherosclerotic lesions may arise from the degeneration and organization of blood clots forming on the inner wall of the blood vessel. The lipid found in such lesions may be derived in part from lipid-rich blood plate lets trapped in the clot. Other findings suggest that when some portion of the inner wall of a blood vessel is deprived of an adequate oxygen supply by such factors as local injury or blood-clot formation, there is an increased uptake of lipoproteins by that portion of the vessel.

NHI Support in This Field

Research exploring the subject of diet in relation to atherosclerosis and coronary heart disease has been productive, as the findings cited above indicate. The growing fund of factual knowledge that is taking shape in this field has resulted in great measure from the research support provided by the National Heart Institute. Literally hundreds of grant-aided research studies in this country and others abroad have contributed basic, clinical, and epidemiological findings.

Investigations in the disciplines of biochemistry, nutrition, and pathology have been particularly fruitful. Many obscure areas and many unsolved problems remain, nonetheless. While some relationships have been clearly demonstrated (e.g., that the fat content of the diet can influence serum cholesterol levels), others have not as yet been proved (e.g., that high cholesterol levels are causally related to more rapid progression of atherosclerosis). Past results suggest that continued investigation will eventually define the precise role of diet in arteriosclerosis and coronary disease.

Diet studies are, of course, only one facet of Heart Institute-supported research aimed at discovering the basic etiology of heart attacks. *The number and variety of factors implicated in atherosclerosis and its consequences, and the strong relationships that some of these seem to have, make it unlikely that this disease has a single cause.* It appears that there may be a multiple etiology, and that the development of atherosclerosis and coronary disease may be owing to the combined or accumulated effects of a diversity of factors.

Among factors that may play significant roles in relation to coronary disease are heredity, hypertension, lipid metabolism, emotional stress, age, smoking, physical activity, blood vessel trauma, vascular anatomy, sex, and obesity. The National Heart Institute supports investigations relating to all of these areas and also supports many studies, ranging from fundamental work in cardiovascular physiology to clinical trials of techniques and therapeutics, from which new information of significance constantly develops.

Excerpts from Symposium on Chemical Carcinogenesis

H.F. Kraybill, PhD
Bethesda, MD, National Cancer Institute

Part II. Carcinogenesis associated with foods, food additives, food degradation products, and related dietary factors.

Since the genesis of tumors may be influenced to a lesser or greater degree by the nutrient environment furnished the host, frequent reference is made to the role of nutrition and dietary factors in carcinogenesis. A current and comprehensive review of nutrition and cancer has been presented by Tannenbaum.[67]

While the obvious association of cancer and nutrition was recognized earlier, a comprehensive assessment of the role of food and diet in the induction of neoplasms must more accurately be viewed in relation to the ingestion of food additives, food contaminants, processing degradation products, and other dietary components.[8,74]

Rapid advances in modern food technology have focused attention on dietary components other than essential nutrients such as food additives and processing degradation products. The utilization of agricultural chemicals as soil fumigants, plant growth regulators, and pesticides that may remain on fruits and vegetables if not adequately removed also contributes food contaminants to the diet of man.

More recently, the nuclear detonations in weapons-testing programs as well as the expanded industrial and medical applications of radioisotopes have introduced radionuclides into the diet as food contaminants. While many of these materials and identified chemical compounds appearing either as additives, contaminants, or degradation products may not be labeled as poisons toxicologically, their latent effect must be assessed in terms of their potential contribution to induction of cancer.

The continuous introduction of synthetic materials as coating materials for films, packages, and containers for foods presents certain problems in terms of migration of these substances from the wrapper or container into the raw or processed food. Whereas some compounds are essential in processing and for improvement of flavor and texture, other organic compounds are added strictly for coloring purposes and enhancement of acceptability. These food colors, coating materials, and a wide spectrum of other materials must be tested thoroughly for their toxic or carcinogenic properties prior to general use.

Accordingly, comprehensive studies dealing with route and rate of absorption, levels of storage in the tissues, and ultimate metabolic fate are now requisite in order to elucidate the mechanism of biologic action of the dietary component under investigation. A comprehensive treatment of this phase of the problem is reviewed in other reports. A general discussion of principles is appropriate, however, before description of the series of observations reported on the genesis of neoplastic disease in animals and man mediated through the multiple factors associated with ingested materials.

Influence of additives, contaminants, and degradation products in foods on mutagenicity and carcinogenicity. A consideration of food intake in carcinogenesis is not restricted to nutrition per se but must also include additives, contaminants, or processing degradation products, which may play a more important role in tumor induction.

Inevitably in the processing of food for preservation and increased palatability, certain products are formed such as the hydroperoxides in autoxidation or heating of fats because of the action of oxygen on unsaturated lipids. These lipid peroxides, or organic peroxides, may act as inhibitors of catalase or peroxidases, and on this mechanism rests a possible explanation of the potential carcinogenic effect.

Radiation, of course, produces effects comparable to autoxidation or polymerization through heating, and it is not surprising that radiation preservation produces end products in food comparable to other processing procedures.

The hydroperoxides are readily absorbed by the organism and soon after ingestion can be detected in anatomic sites, such as the liver or adipose tissues of the muscles. These organic peroxides may attack vitamins, inhibit certain enzymes, affect the mitochondria, interfere with the function of cortisone, reduce the viscosity of desoxyribonucleic acid, attack hemoglobin, and destroy vital sulfhydryl linkages. At significant levels, they will, as a result of vitamin destruction, produce sterility in rats and dogs (vitamin E destruction) and hemorrhagic diathesis in rats (vitamin K destruction).[30,37]

Organic peroxides may produce cleavage in the nucleic acid chain resulting in alteration in desoxnibonucleic acid through depolymerization, thus accounting for their mutagenic action in Drosophila, Neurospora, and Aspergillus.[61] These effects are potentiated in some cases by the addition of certain carbonyls, particularly formaldehyde.

Specific references to food additives, food contaminants, and food degradation products in carcinogenesis:

Food colors and additives. The basis for acceptance or rejection of these substances applied by the food industry has been largely through extensive animal experimentation.

Whereas the critical appraisal is on the basis of oral feeding, and certainly a proved carcinogen by this technique would be rejected by Food and Drug authorities, if tumors are demonstrated by skin painting or injection, this may also be the basis for rejection.

Auramine O and Tetramethyl diamino-diphenyl-cetonimine are representative of carcinogenic food colors, and the component *B*-naphthylamine in yellow OB and AB or other impurities in this color may be carcinogenic. In the Union of South Africa, according to Steyn,[63] nigrosine and benzopurpurine have been removed from permitted list of dyestuffs on the basis of toxic effects and not at the moment because of tumorigenic incidence.

Since carbon blacks have been shown to contain 3,4-benzpyrene as a contaminant, these materials, along with activated charcoals used as food colors, have been investigated. The petroleum waxes used in food packaging and as food additives (chewing gum) have been and are receiving some investigation, since certain petroleum fractions have been shown to be highly carcinogenic.[19]

In feeding rats D&C red No. 9 for two years, Davis and Fitzhugh[11] found no apparent effect on the growth rate, mortality, and occurrence of tumors from this food color.

Dulcin or *p*-phenetylurea (Valzin or Sucrol) used as sweetening agent has been shown to be toxic and hepatomatous to experimental animals.[18] Fitzhugh[18] compared the chronic toxicities of P-4000 (2-amino-4-nitrophenylpropyl ether), cyclamate sodium, and saccharin, and on these tests, Dulcin was banned by the U.S. Food and Drug Administration as a food sweetening agent.

For fattening of poultry and cattle, diethylstilbestrol has been used as implanted pellets and as an additive to rations and has been shown to have carcinogenic properties.[21] The fact that a tissue residue remains in poultry from pellet implantation has resulted in prohibition of its use, but in livestock feed it has not been discontinued in the United States.

More recently, attention has been focused not on the role of nutrition but rather on the specific and additive influences of perhaps more important contributors, such as the materials that are added to food (in processing), contaminants such as spray residues, and finally, through processing, canning, or heating, the degradation products that are ever present.

It is not surprising with the present state of the art and advances in food research and technology that over 800 chemical substances find their way into foodstuffs. Many of the chemical additives such as emulsifiers and stabilizers for aqueous and fat phases are designed to facilitate processing of many food products. Others enhance color, improve palatability, extend shelf life, and modify texture.

To prevent insect damage to fruits and vegetables and to control size of plants, pesticides and plant growth regulators have become an integral part of agricultural practice. Spray or plant residues of these chemicals may not be completely removed in some cases prior to consumption and hence contribute to food contamination.

[*Editor's note*: References for this article are not available in original document.]

Dental Caries and the Pediatrician

Editorial by W.C. Black, MD. Reprinted from *American Journal of Diseases of Children*, Vol. 106, No. 2, August 1963.

Dental caries, the most common "chronic disease" of American children, is primarily caused by improper diet. Heredity plays a secondary role. The dietary fault has been shown to be refined carbohydrate. Although it is unlikely that the pediatrician's admonitions will correct the fault and change our national food habits, still it is clearly his responsibility to make sure that his patient's parents *know* that refined carbohydrate is the cause of nearly all caries and to encourage a proper physiologic diet.

Although it has not been shown that the daily ingestion of minute amounts of fluoride ion is physiologic and necessary for normal dental health, still it is clearly true that even in the presence of a caries-producing diet, small daily amounts of fluoride ion exert a strong prophylactic action between the fifth month of gestation and the age of nine or ten years. The fluorine is incorporated into the enamel of the teeth, rendering them much less susceptible to decay.

The dose of fluoride ion necessary to induce this prophylactic effect is approximately one mg per day. Larger amounts permanently stain the enamel various shades of yellow, tan, and brown.

Of the various ways to administer prophylactic fluorine, the method of regular exact dosage is obviously best. Fluoride ion can be added (without expense) to vitamins, to canned milk or formulas for infant and child consumption, or to any other item of diet taken regularly in the same amount daily.

If this prophylaxis is to be employed during pregnancy, it can be added (without expense) to prenatal capsules, cow's milk, calcium tablets, or any other item of diet regularly consumed in fixed amounts by the pregnant woman.

Now that the proper dose of fluorine for dental caries prophylaxis has been determined, it is unnecessary and unwise to wastefully add it to community water supplies for the following cogent reasons:

1. Dosage is highly variable and inaccurate.
2. Older children and adults need not and should not be dosed with the drug.
3. Fluoridated water is of no benefit to plants and *may* be undesirable for edible plants when such plants are ingested by animals, birds, or humans.
4. Fluorine is a potent poison.

W.C. Black, MD
College Ave Professional Bldg.
4529 College Ave
San Diego 15, Calif.

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Toll Free: +1 (866) 407-9323
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PO Box 270091, Fort Collins, CO, 80527
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